The Journal of HEREDITY

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X-Ray Resusciation of Heat-Killed Seeds

Keratosis and Friedreich's Ataxia Combined in a Single Pedigree

Interaction of Color Genes in Beans

Living Sextuplets and Octuplets — Fact or Fiction?

VOLUME 39 JULY, 1948 NUMBER 7

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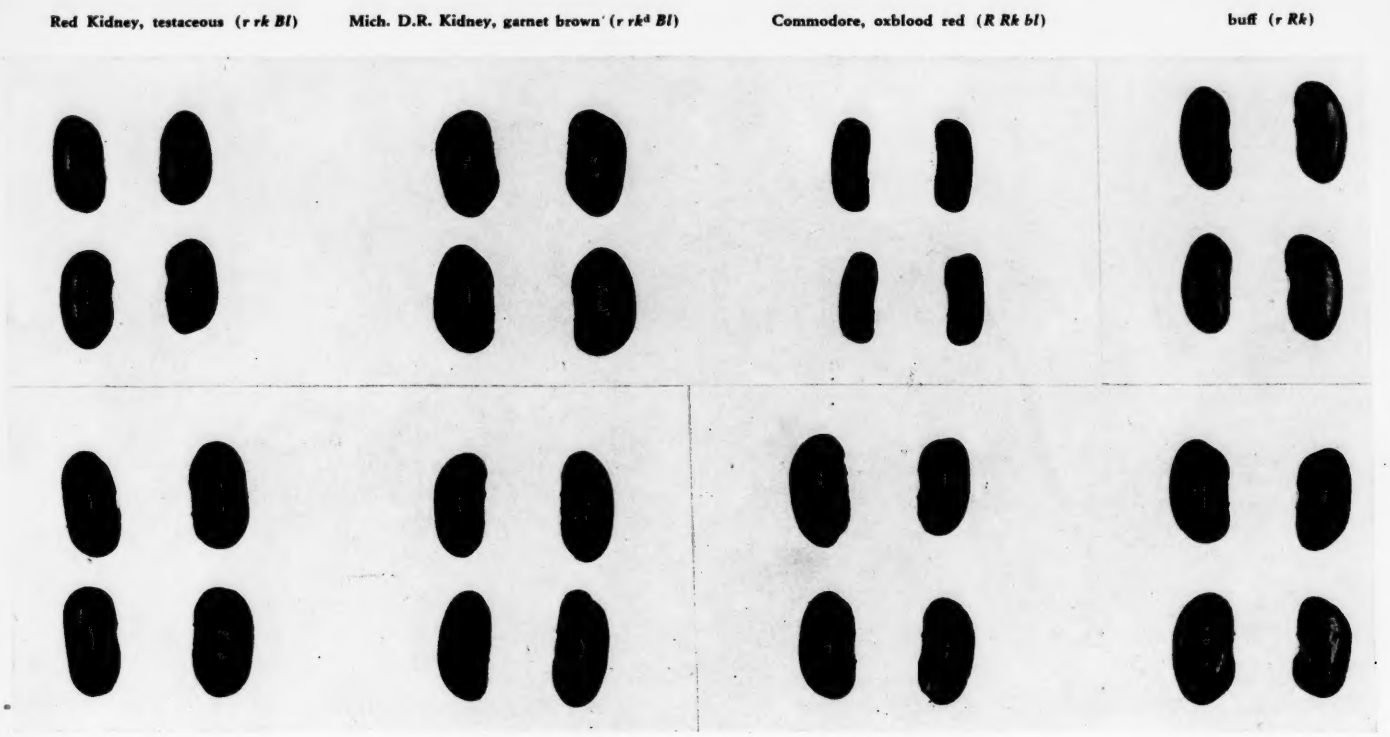
Red Kidney, testaceous (r rk Bl)
Mich. D.R. Kidney, garnet brown (r rk^d Bl)
Commodore, oxblood red (R Rk bl)
buff (r Rk)
Indian purple (R Bl)
Indian purple/buff (Rr Rk Bl)
Oxblood red/buff (Rr Rk bl)
Oxblood red/testaceous (Rr rk bl)

INTERACTION OF THREE SEED COAT COLOR GENES IN THE BEAN

Figure 1

In the bean two independently inherited genes, *R* and *Rk*, condition red seed coat color. A third gene, *Bl*, modifies oxblood red to purple, but does not affect testaceous. Allels of these three genes interact to produce the eight different color patterns illustrated here.

SEED-COLOR INHERITANCE IN BEANS

Interaction of the Alleles at the *R*, *Rk* and *Bl* Loci in *Phaseolus vulgaris*

FRANCIS L. SMITH AND CATHARINE BECKER MADSEN
Division of Agronomy, University of California

THE three genes whose interaction is the object of this report have been reported in earlier literature. The *R* symbol was first applied by Tjebbes and Kooiman[8] to a gene for red seed coat color. Lamprecht[2] has demonstrated that *R* produces mottling when heterozygous. Later[3] he found seven alleles of the *R* gene, one of which is the mottling gene *M* which he gave the symbol R^{ma}. In an earlier paper,[5] the senior author reported the interactions of the *R* gene and another red seed coat color gene to which he gave the symbol *Rk* (from the Red Kidney variety). In the presence of *R R*, the *Rk* gene had no effect. The interaction of *R* and *Rk* was most striking when the *R* gene was heterozygous; the genotype *R r Rk* was mottled on a buff background, and *R r rk rk* was mottled on a testaceous background. In beans of the genetic constitution *r r Rk Rk* the seed coats were buff colored, while beans of the constitution of *r r rk rk* were testaceous in color. Identical reactions were shown for *Rk* in the presence of the mottling gene now called R^{ma}. This constant mottling gene occurs in many commercial varieties, whereas the inconstant mottling of heterozygous *R r* cannot be fixed in a variety.

The *Bl* symbol was first used by Tjebbes and Kooiman[8] to designate a modifier which changes *R r* red beans to purple. The senior author[5] used the same symbol to describe an apparently identical gene which changes *R R* red beans to purple, and also changes the color of mottling from red to purple in the presence of either *R r* or R^{ma}.

The phenotypic expression of the interactions of these three genes then can be seen in the seed coat (see Figure 1). Colors of the various genotypes are as follows (a single gene symbol implies homozygosity of that locus):

R R Rk Bl or *R R rk Bl* — purple
R R Rk bl or *R R rk bl* — oxblood red
R r Rk Bl — purple/buff
R r Rk bl — oxblood red/buff
R r rk Bl — purple/testaceous
R r rk bl — oxblood red/testaceous
r r Rk Bl or *r r Rk bl* — buff
r r rk Bl or *r r rk bl* — testaceous

Lamprecht's recent paper[3] reviewed the earlier paper by the senior author.[5] However, Lamprecht missed the significance of the *Rk* gene. He went to great length reorganizing data from tables to show that the action of *Rk* and *rk* was always in the presence of *r r*, and stated that this proved the nonexistence of *Rk* and recommended that "it should be deleted from the literature." Lamprecht says that *r r* without other color genes is colorless. To this, the authors have no objection. But *r r* with *Rk* produces buff colored beans and *r r* with *rk* produces testaceous beans. Further studies of these gene interactions are reported below.

Materials and Methods

Two red-seeded varieties heretofore untested were used in these studies. One was Michigan Dark Red Kidney obtained twice from the Michigan Agricultural Experiment Station and carried under the accession numbers 8099 and 8346. A third accession of this variety was obtained from Canada and carried as 8795. All three were identical in seed coat color, namely, garnet brown. The second variety, which has an oxblood red seed coat, was Commodore, obtained from the Associated Seed Growers and grown under accession number 8353. These varieties were crossed with color tester stocks which are listed in Table I.

The colors of the F_1 and F_2 generations were matched and classified with Ridgway's Color Standards.[4]

For brevity, the Ridgway color "Pink-

ish buff" is called simply buff and "Indian purple" is abbreviated to purple. Since a number of dark red colors were encountered, Ridgway's Garnet brown, Oxblood red, Dragon's blood red, and testaceous are used to distinguish beans of different colors and genetic constitutions. Mottled beans are written as fractions, the mottling color being the numerator and the ground color the denominator. The observed results of the crosses were tested with theoretical results by the χ^2 method and values of P were obtained from Fisher's[1] tables.

Results

Crosses involving Commodore

Commodore was crossed with a number of known color genotypes. One of these was Buff 4802, *r r Rk bl*. The F_1 plants were red/buff and the F_2 segregated 1 oxblood red: 2 oxblood red/buff: 1 buff as shown in Table II. This indicates that only the red gene, which causes mottling when heterozygous, was involved. Commodore therefore must have the same *Rk* and *bl* gene as the tester line, and must differ from the tester in having *R*. Its genotype then is *R Rk bl*.

When Commodore was crossed with testaceous 4812 (*r rk bl*) the F_1 was red/buff, and the F_2 segregated for both *R r* and *Rk rk* as shown in Table III. There was no segregation at the *bl* locus, all F_2 plants showing *bl* rather than *Bl*. Since the tester contributed *r rk bl*, Commodore must have contributed *R Rk bl*. This confirms the results of the previous cross.

In order to test Commodore with the red mottling gene, R^{ma} it was crossed with red/testaceous 4801, R^{ma} *rk bl*. The F_1 plants were red/buff and the F_2 segregated for R^{ma} and *Rk* as shown in Table IV. The *P* value was very low for the dihybrid ratio; however, the segregation of *R* R^{ma}, the genes of interest in this cross, gave a close fit to a 3:1 ratio with a *P* value of .90-.80. This confirms Lamprecht's[3] hypothesis that *R* and R^{ma} belong to the same allelomorphic series. Since *Rk* was segregating and no effects of *Bl* were apparent, these results are in conformity with the others which give the genetic constitution of Commodore as *R Rk bl*.

When Commodore was crossed with testaceous 4803 known to be of the constitution *r rk Bl*, the effect of the *Bl* gene was shown by the purple/buff color of the seed coat of the F_1. Accordingly, this cross was expected to segregate for all three genes. The results shown in Table V indicate a trihybrid segregation, although the fit to theoretical ratios was rather poor with a *P* value of .01. The *P* values for the segregation of each gene separately are: *R r*, very low; *Rk rk*, .05 to .02; and *Bl*, .05 to .02. Although these data do not fit expectation closely, they are not contradictory to the hypothesis that Commodore is genotypi-

TABLE I. Color variants used as testers.

Color	Accession number	Formula	Derived from a cross
Oxblood red/testaceous	4801	R^{ma} *rk bl*	White Kidney × Nagazura 34.160
Buff	4802	*r Rk bl*	do
Testaceous	4803	*r rk Bl*	do
Testaceous	4805	*r rk Bl*	do
Testaceous	4812	*r rk bl*	Dark Red Kidney × Red Kidney 34.167
Buff	4813	*r Rk bl*	do
Oxblood red	4814	*R Rk bl*	do
Testaceous (California Red Kidney)	7811	*r rk Bl*	Well's Red Kidney × Nagazura[6]

TABLE II. F_2 segregation of buff 4802 *r Rk bl* × Commodore 8353, oxblood red *R Rk bl*,* and reciprocal.†

Color	Formula	Ratio	Obs.	$(O-C)^2/C$	P
Oxblood/buff	*R r Rk*	2	375	.024	
Oxblood red	*R R Rk*	1	207	2.371	
Buff	*r r Rk*	1	162	3.097	
Total			744	5.492	.10-.05

*Crosses 43.107, 108, 109, and 110; 15 red/buff F_1 plants.
†Crosses 43.142 and 143; nine F_1 plants.

TABLE III. F_2 segregation of Commodore 8353 oxblood red *R Rk bl* × testaceous 4812 *r rk bl*.*

Color	Formula	Ratio	Obs.	$(O-C)^2/C$	P
Oxblood red/buff	*R r Rk*	6	42	1.251	
Oxblood red/testaceous	*R r rk*	2	26	5.323	
Oxblood red	*R R Rk* and *rk*	4	35	.087	
Buff	*r r Rk*	3	26	.049	
Testaceous	*r r rk*	1	4	2.228	
Total			133	8.938	.10-.05

*Crosses 42.104, and 105; two red/buff F_1 plants.

TABLE IV. F_2 segregation Commodore 8353 *R Rk bl* × red/testaceous 4801 R^{ma} *rk bl*.*

Color	Formula	Ratio	Obs.	$(O-C)^2/C$	P
Red/buff	R^{ma} *Rk*	9	58	5.213	
Red/testaceous	R^{ma} *rk*	3	45	13.765	
Oxblood red	*R Rk* and *rk*	4	36	.045	
Total			139	19.023	very low

*Crosses 42.102, 42.103; two red/buff F_1 plants.

cally *R Rk bl*. This constitution has been verified by crossing with four different genetic stocks.

Crosses involving Michigan Dark Red Kidney

This variety was crossed with testaceous lines carrying *bl* or *Bl*, with buff, and with oxblood red tester stocks.

In Table VI the results from a number of crosses of Michigan Dark Red Kidney × testaceous are summarized. In some crosses, the testaceous tester parent had been previously determined to be of the constitution *r rk Bl* and in others it was *r rk bl*. As is shown in Table VI the F_1 plants and one half of the F_2 population were slightly darker than testaceous and were described as Dragon's blood red. Only one gene was segregating in these crosses. There was no discernible difference between crosses made with testaceous carrying *Bl*, and testaceous carrying *bl*. The Michigan Dark Red Kidney color, garnet brown, is not affected by the *Bl* gene. In this respect it is like the testaceous *rk* gene.

Michigan Dark Red Kidney was also crossed with two buff lines, 4802 and 4813, both known to be genotypically *r Rk bl*. The F_1 plants were buff and a monohybrid segregation was obtained in the F_2 as shown in Table VII.

The garnet brown of Dark Red Kidney thus differs from the testaceous *rk* and also from buff *Rk* by a single mendelian factor. Since *Rk* and *rk* are allels, the requirements of the so-called triangle test for multiple allels of the same gene have been met, and garnet brown is therefore a third allel of *Rk* buff and *rk* testaceous. Garnet brown may be designated as rk^d for Dark Red Kidney. *Rk* is completely dominant to both other allels, and *rk* is partially dominant to rk^d.

Michigan Dark Red Kidney was tested for the genes *R* and *Bl* by crossing with oxblood red 4814 known to be of the constitution *R Rk bl*. The F_1 plants were purple/buff, and a modified trihybrid segregation for *R r*, *Rk* rk^d, and *Bl bl* was obtained in the F_2 as shown in Table VIII. Since the tester contributed *R Rk bl*, Michigan Dark Red Kidney must carry *r* rk^d *Bl*. The segregation is similar to that obtained by crossing a testaceous bean, such as California Red Kidney *r rk Bl*, with Commodore, *R Rk bl*, an oxblood red, as in Table V, with

TABLE V. F_2 segregation Commodore 8353 *R Rk bl* × testaceous 4803 *r rk Bl*,* and reciprocal.†

Color	Formula	Ratio	Obs.	$(O-C)^2/C$	P
Purple/buff	*R r Rk Bl*	18	306	.146	
Purple/testaceous	*R r rk Bl*	6	125	4.130	
Oxblood red/buff	*R r Rk bl*	6	82	4.749	
Oxblood red/testaceous	*R r rk bl*	2	31	.405	
Purple	*R R Bl Rk* or *rk*	12	244	6.044	
Oxblood red	*R R bl Rk* or *rk*	4	76	.609	
Buff	*r r Rk Bl* or *bl*	12	181	3.627	
Testaceous	*r r rk Bl* or *bl*	4	67	.090	
Total			1,112	19.800	.01

*Crosses 43.113, .114, .115, .116; 13 purple/buff F_1 plants.
†Crosses 43.145, .146, .147, .148, .149; 17 purple/buff F_1 plants.

TABLE VI. F_2 segregation of Michigan Dark Red Kidney garnet brown × Red Kidney and other testaceous tester lines.

	Garnet brown	Dragon's blood red	Testaceous	X^2	P
Ratio	1	2	1		
Michigan Dark Red Kidney* × *r rk Bl*	120	204	113	2.15	.50-.30
Michigan Dark Red Kidney† × *r rk bl*	146	280	133	.71	.70

*Crosses 39.062, 39.065, and 39.084.
†Crosses 38.472, 39.073, and 39.070.

TABLE VII. F_2 segregation of Michigan Dark Red Kidney 8346 × Buff *r Rk bl and reciprocal.†**

Cross	Color	Formula	Ratio	Obs.	$(O-C)^2/C$	P
8346 × 4813*	Buff	*Rk*	3	124	.002	
	Garnet brown	*rk*	1	42	.006	
Total				166	.008	.95-.90
4802 × 8346†	Buff	*Rk*	3	277	1.078	
	Garnet brown	*rk*	1	70	3.234	
Total				347	4.312	.05-.02

*Crosses 42.101; five buff F_1 plants.
†Crosses 43.111, .112, .119, .120 and .121; twelve buff F_1 plants.

TABLE VIII. F_2 segregation of Michigan Dark Red Kidney 8346 × oxblood red 4814 *R Rk bl and reciprocal.†**

Color	Formula	Ratio	Total Observed	Total $(O-C)^2/C$
Purple/buff	*R r Rk Bl*	18	176	.0022
Purple/garnet brown	*R r* rk^d *Bl*	6	63	.2883
Oxblood red/buff	*R r Rk bl*	6	51	1.0546
Oxblood red/garnet brown	*R r* rk^d *bl*	2	12	2.9594
Purple	*R R Bl Rk* or *rk*	12	128	.8922
Oxblood red	*R R bl Rk* or *rk*	4	44	.5748
Buff	*r r Rk Bl* or *bl*	12	115	.6422
Garnet brown	*r r* rk^d *Bl* or *bl*	4	39	.0016
Total			628	
X^2				6.4153
P				.70-.50

*Crosses 38.473; ten purple/buff F_1 plants.
†Reciprocal crosses 43.122, .123, .124, .125; 16 purple/buff F_1 plants.

the exception that in the cross with Michigan Dark Red Kidney, the background color of mottled beans and the color of *r r* self-colored beans is garnet brown instead of testaceous. The only difference, then, between *rk* and rk^d is a substitution of garnet brown for testaceous whenever rk^d is used in crosses. It is interesting to note that both California Red Kidney and Michigan Dark Red Kidney carry the *Bl* gene.

Discussion

It is apparent, from the results of the foregoing seven crosses as well as from earlier work of the senior author, that testaceous and its allels form a series which segregate independently of the *R* red series, and of the *Bl* purple modifier. The *Rk* series has also been shown[7] to be loosely linked with *Br*, a brown gene affecting the mottled areas in the Pinto variety.

All these sources of genetic evidence seem ample to substantiate the presence of *Rk* and its allels in beans; therefore Lamprecht's recommendation[3] to "delete it from the literature" must be rejected.

Summary

The interactions of eight seed coat color genes were studied and the genes were found to belong to three independent allelomorphic series: *R*, R^{ma}, and *r*; *Rk*, *rk*, and rk^d; and *Bl* and *bl*.

The *R* gene, when homozygous dominant, makes the seed coats oxblood red; when heterozygous, it makes them oxblood red mottled on a ground color determined by other genes; when homozygous recessive, the color is entirely determined by other color genes. In contrast, R^{ma} produces a true red mottling whether homozygous dominant or heterozygous. The *r* allel has no effect on color.

The *Rk* series has three known allels, *Rk* buff, *rk* testaceous, and rk^d garnet brown. The rk^d allel was found in Michigan Dark Red Kidney and is reported for the first time in this paper. In order of dominance, these allels are *Rk*, *rk*, rk^d.

Trihybrid ratios involving the three gene loci are modified by the following interactions.

Bl modifies the red color of *R* or R^{ma} to purple but has no effect in the presence of *r r*. It has no effect on the *Rk* series of allels. The homozygous dominant *R* is epistatic to all genes in the *Rk* series. The *Rk* series determines the color of beans homozygous for *r r*, and the ground color in beans mottled by either R^{ma} or heterozygous *R r*.

Commodore was found to have the color genes *R Rk bl*, and Michigan Dark Red Kidney was found to contain *r* rk^d *Bl*.

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RESUSCITATION OF HEAT-INACTIVATED SEEDS WITH X-RADIATION*

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THIS is a preliminary report on experiments to determine the effect of pre- or post-irradiation on seeds, in combination with a barely lethal heat treatment. Remarkable results were obtained in a series of tests with barley and einkorn. Seeds which were exposed to a lethal heat treatment could be "resuscitated" by subsequent exposure to X-radiation; and likewise, some doses of X-radiation were capable of "protecting" seeds from subsequent lethal heat treatments. Muller[6] recently reiterated the view held by many that "a large proportion if not the great majority of the somatic effects of irradiation . . . arise secondarily as consequences of genetic effects," so the findings reported herein have obvious genetic interest and significance.

Methods and Results

In one series of experiments with barley, 250 seeds were handled in the following manner: 1) 50 untreated seeds were used as a control; 2) 50 seeds were given 12,000*r* units; 3) 50 seeds were subjected to a barely lethal heat treatment of 70°C. for 30 minutes; 4) 50 seeds were given 12,000*r* units and then immediately subjected to 70°C. for 30 minutes; 5) 50 seeds were subjected to 70°C. for 30 minutes and then immediately given 12,000*r* units. The experiment was repeated with four additional 250-seed lots. The X-ray dosage was progressively increased in succeeding series to 18,000*r* for the second; to 24,000*r* for the third; to 30,000*r* for the fourth; and to 36,000*r* for the fifth series. The X-ray set was operated at 27 K.V.P. and 15 milliamps, giving approximately 1,000*r* per minute at the surface of the seeds.

The barely lethal heat treatment was determined by subjecting 50-seed samples to temperatures which increased by intervals of 5°C. from 60°C. to 85°C. The lowest temperature which completely inhibited germination was used in studies of the effects of combination heat and X-ray treatments. The balance between killing and survival was rather

TABLE I. Survival and growth of dormant seeds of barley given various doses of irradiation before and after a lethal heat treatment.

Treatment	No. of seeds	Survival (%)	Height (inches)
I.			
1. Control	50	98	4
2. 12,000*r*	50	96	3.5
3. 70°C. for 30′	50	0	0
4. 12,000*r* + 70°C. for 30′	50	0	0
5. 70°C. for 30′ + 12,000*r*	50	100	3.75
II.			
1. Control	50	100	3.75
2. 18,000*r*	50	94	2.5
3. 70°C. for 30′	50	0	0
4. 18,000*r* + 70°C. for 30′	50	0	0
5. 70°C. for 30′ + 18,000*r*	50	94	3.0
III.			
1. Control	50	100	3.75
2. 24,000*r*	50	96	2.0
3. 70°C. for 30′	50	0	0
4. 24,000*r* + 70°C. for 30′	50	90	2.0
5. 70°C. for 30′ + 24,000*r*	50	92	2.0
IV.			
1. Control	50	100	3.75
2. 30,000*r*	50	92	1.75
3. 70°C. for 30′	50	0	0
4. 30,000*r* + 70°C. for 30′	50	2	1.0
5. 70°C. for 30′ + 30,000*r*	50	90	2.0
V.			
1. Control	50	100	3.5
2. 36,000*r*	50	68	1.0
3. 70°C. for 30′	50	0	0
4. 36,000*r* + 70°C. for 30′	50	72	1.0
5. 70°C. for 30′ + 36,000*r*	50	96	1.75

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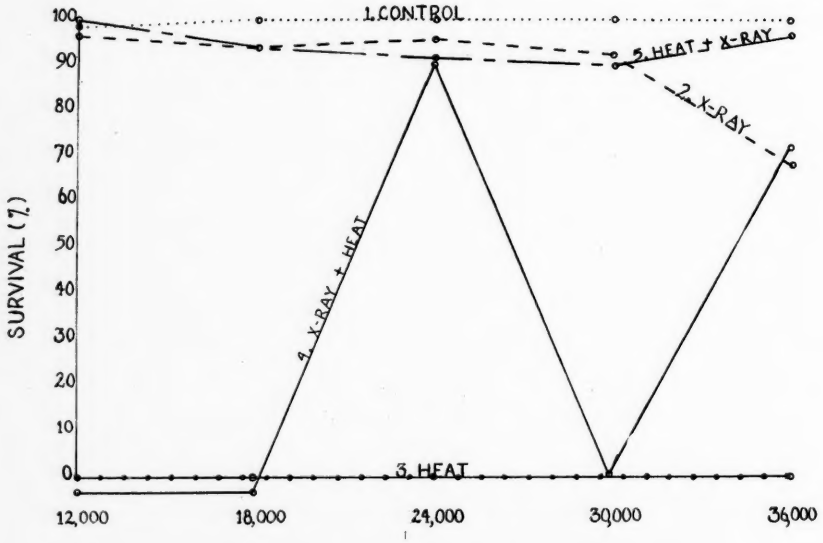
†The writers wish to thank S. T. Stephenson, Professor of Physics, and H. J. Dana, Director of the Engineering Experiment Station, for generous cooperation in making available X-ray equipment and other facilities used in this study.



EFFECTS OF HEAT AND X-RAYS ON SEED SURVIVAL

Figure 2

Graph showing effects of certain doses of X-rays in preventing heat-inactivation (curve 4), and resuscitation of heat-inactivated seeds by a considerable range of X-ray dosages (curve 5). The curves for untreated (curve 1) and heat-treated (curve 3) comprise seed samples which received no radiation. The sample represented by curve 2 was X-rayed but not heat treated.

delicate, because usually there was almost 100% survival at a temperature 5°C. below the temperature used in the tests. Possibly a factor in this uniformity of response to heat was the fact that the seeds were selected for uniform size, heredity, and history.

Data obtained from the five trials conducted with barley are presented in Table I, and shown graphically in Figure 2. In all five trials 90% or more of the seeds apparently killed by heat treatment, and then immediately X-rayed, survived. In most cases, particularly with heavier doses of X-rays, seeds treated with heat and then X-rayed grew somewhat better than seeds given X-radiation alone.

Seeds which were X-rayed before they were subjected to the lethal temperature responded somewhat differently than did seeds given the same treatments in the reverse order. At some X-ray dosages there was no protection from the heat inactivation, whereas at other dosages there was almost as complete a survival as there was in those lots in which seeds were subjected to heat before they were X-rayed. The protection phenomenon was evidently more dependent on X-ray dosage when the heat treatment succeeded the irradiation. The results reported herein were substantiated by another complete series of treatments involving 1,250 seeds in which the same responses at approximately the same dosages were obtained, and by four other incomplete series of tests involving 3,500 seeds in which all essential aspects of the responses were obtained which were necessary to show that "resuscitation" or "protection" actually occurred.

Einkorn Series

A series of treatments similar to those conducted with barley were applied to five 250-seed lots of einkorn. Each series consisted of the following: 1) 50 untreated seeds were used as a control; 2) 50 seeds were X-rayed; 3) 50 seeds were subjected to a barely lethal heat treatment of 75°C. for 30 minutes; 4) 50 seeds were X-rayed and then immediately subjected to a temperature of 75°C. for 30 minutes; 5) 50 seeds were subjected to a temperature of 75°C. for 30 minutes and then immediately X-rayed. In the first series the X-ray treatment was 6,000*r*, and the dosage was increased by intervals of 4,000*r*, to 22,000*r* for the fifth series. The data from these trials with einkorn are presented in Table II. These data and the data from Table I indicate that barley and einkorn responded in the same way to similar combined heat and X-ray treatments, but at different levels of dosage.

Discussion

Numerous investigators* have shown that heat modifies both micro- and macroscopic biological effects of X-radiation. However, the authors are not aware of any data showing the modification of heat effects by the use of X-rays. The fact that so little is known about the specific effect of heat and X-rays on living systems makes it difficult to perceive what causes the interaction, which in these tests resulted in resuscitation of heat-inactivated seeds, or the protection of seeds from heat-inactivation.

TABLE II. Survival and growth of dormant seeds of einkorn given various doses of irradiation before and after a lethal heat treatment.

Treatment	No. of seeds	Survival (%)	Height (inches)
I.			
1. Control	50	100	5
2. 6,000*r*	50	80	2.5
3. 75°C. for 30′	50	0	0
4. 6,000*r* + 75°C. for 30′	50	0	0
5. 75°C. for 30′ + 6,000*r*	50	90	3
II.			
1. Control	50	100	5
2. 10,000*r*	50	76	1
3. 75°C. for 30′	50	0	0
4. 10,000*r* + 75°C. for 30′	50	80	1.5
5. 75°C. for 30′ + 10,000*r*	50	82	2.0
III.			
1. Control	50	96	5
2. 14,000*r*	50	60	0.5
3. 75°C. for 30′	50	6	3
4. 14,000*r* + 75°C. for 30′	50	0	0
5. 75°C. for 30′ + 14,000*r*	50	56	0.5
IV.			
1. Control	50	100	5
2. 18,000*r*	50	40	0.5
3. 75°C. for 30′	50	0	0
4. 18,000*r* + 75°C. for 30′	50	44	0.5
5. 75°C. for 30′ + 18,000*r*	50	40	0.5
V.			
1. Control	50	100	5
2. 22,000*r*	50	24	0.5
3. 75°C. for 30′	50	4	3.0
4. 22,000*r* + 75°C. for 30′	50	0	0
5. 75°C. for 30′ + 22,000*r*	50	15	0.5

*See Literature Citations 1-5 and 8-10.

Death from heat has been variously attributed to divers effects, among them: 1) coagulation of protein components of protoplasm; 2) changes in the physical state of lipids; and 3) destruction or inactivation of enzymes. At present the theory concerned with coagulation of protein components of protoplasm seems to be most generally accepted. Coagulation is considered to be an irreversible reaction. It is necessary, therefore, if the killing effect of heat is an irreversible coagulation phenomenon, to assume that by some means, as yet unknown, X-rays inhibit or prevent coagulation resulting from exposure to heat. The data presented for the treatments in which heat preceded X-radiation could well be interpreted on this premise. However, the relationship between resuscitation and dosage, in the tests in which X-radiation preceded heat treatment makes it improbable that this interpretation is the correct one. If it were merely a matter of altering the coagulation effect of heat, there presumably would be a threshold dose of X-rays above which the irradiation would be adequate to prevent the coagulation. However, the different results obtained when X-radiation preceded heat treatment as compared with the results when X-radiation followed heat treatment, and the lack of correlation between resuscitation and X-ray dosage, indicate that such was not the case.

In a study of X-ray effects on the protoplasm of Spirogyra, Northern and MacVicar[7] found that the "structural viscosity" decreased at first with lower dosages, but increased with longer ex-

posure, becoming in some instances greater than the viscosity in control filaments. In some experiments they observed that the increase in viscosity was followed by a second decrease. In one test they believed the second decrease was probably followed by a second increase in viscosity.

There appears to be a decided similarity in the up and down trends in the survival of seeds that were X-rayed and then heated, as reported herein, and in the variations in viscosity noted by Northern and MacVicar.

In order to explain the up and down trends of curve 4 in Figure 2, it must be surmised either that a single substance or reaction varied with dosage of X-rays or that a second substance or reaction developed at a different rate to counteract the effect of the first substance or reaction. The substance or reaction could be any one of a number of things resulting from ionizations. In other words, some effect other than simple coagulation of protein seems to be a factor in causing death from heat.

If the killing effect of heat is attributed to changes in the physical state of lipids or to the destruction or inactivation of enzymes, it is again necessary to assume that X-rays either: 1) prevented the changes in these systems from reaching a climax, or 2) aided in their reconstruction or revival once they had been destroyed or inactivated. At present there is insufficient information for a decision as to what the mechanism of interaction of combination heat and X-ray treatments is.

Summary

1. X-raying dormant seeds of barley immediately after they had been given a "lethal" heat treatment resulted in the resuscitation of 90% or more of them. This result was obtained over a considerable range of X-ray dosages.

2. Seeds which were X-rayed *before* they were given a "lethal" heat treatment were protected from heat-inactivation by some relatively low and high X-ray dosages but not by intermediate dosages.

3. The mean survival of barley seeds given a "lethal" heat treatment and then X-rayed was greater than the mean survival of seeds which were given X-radiation only. In this sense heat treatment also protected seeds from irradiation, just as in some other combinations of treatments, X-radiation protected the seeds from injury by heat.

4. The data presented do not appear to substantiate the hypothesis that the killing effect of heat is due simply to coagulation of protoplasmic proteins.

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KERATOSIS AND FRIEDREICH'S ATAXIA

In the Same Sibship

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HEREDITARY keratosis palmarum et plantarum involves an excessive thickening of the skin of the palms and soles. The condition was given this name by Unna[5] although it was originally described by Thost in 1880, as quoted by Unna. Cockayne[3] lists the following additional names for the abnormality: tylosis palmarum et plantarum, keratosis palmare et plantare, icthyosis palmarum et plantarum, keratosis palmaris et plantaris, and Unna-Thost's disease. In the interest of brevity, keratosis in the following discussion means Unna's keratosis palmaris et palmarum. In most pedigrees keratosis is dependent upon a dominant gene, as Cockayne in his extensive review of the literature up to 1930 shows. Chung[2] traced this dominant defect through six generations in one Chinese family and seven generations in another Chinese family. A Greek family studied by Bloom, quoted by Gates[4] also showed dominant inheritance of the trait.

The inheritance of Friedreich's ataxia, a well known condition in which lesions of the spinocerebellar tracts occur, has been studied by a number of authors. A more severe autosomal recessive form and a milder dominant form of Friedreich's ataxia are recognized.[1]

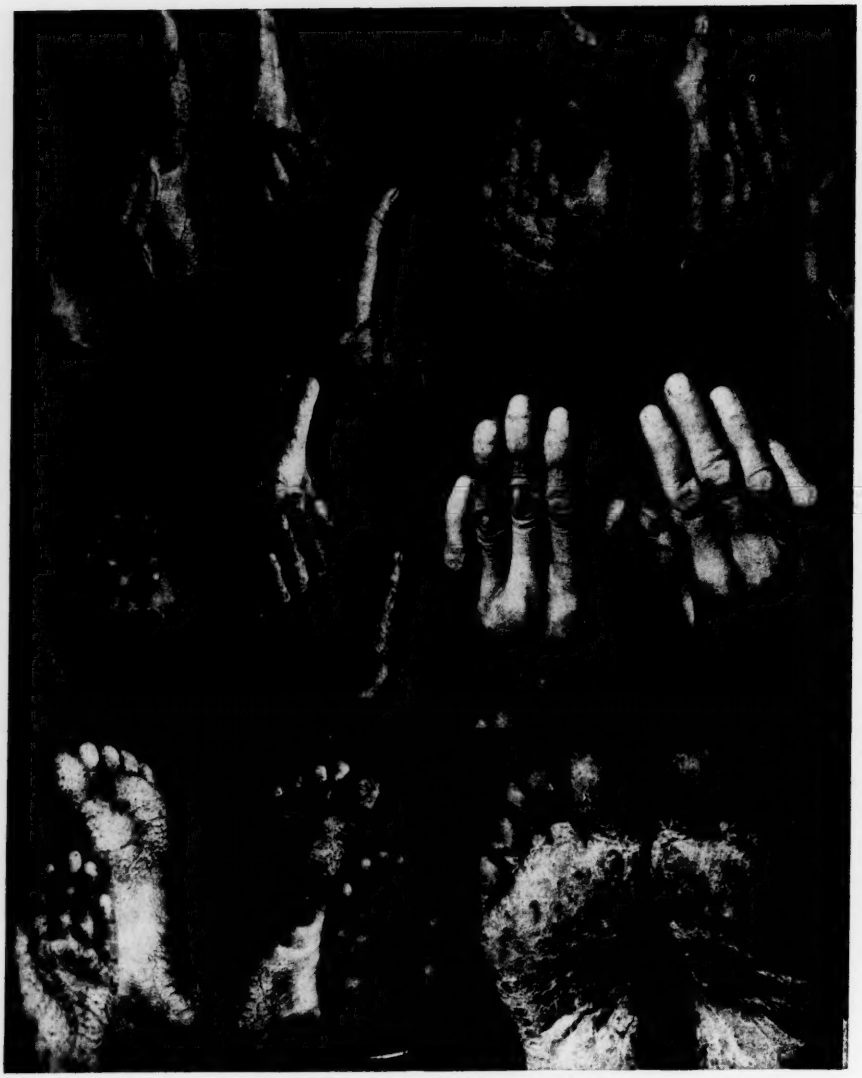
The purpose of the present study is to describe a new pedigree in which keratosis and/or Friedreich's ataxia occurred in certain members of an Arab family.

The propositus of the present pedigree, IV-12, suffers with both keratosis and Friedreich's ataxia. The thick keratosis covers the palms of the hands to a point just proximal to the wrist flexure and also the soles of the feet, as the photographs in Figures 3*D* and 3*F* show. Deep creases in palms and soles appear as red canals in the thickened yellow epidermis. The propositus states that patches of the horny epidermis are cast off occasionally from palms and soles at different times throughout the year. Keratosis in the three other affected members of generation III and IV closely resembles the condition in IV-12, the propositus. Figures 3*A*, 3*B*, 3*C*, 3*D*, 3*F* show the rough surface resulting from the sloughing off of small and large areas of epidermis of palms and soles in different affected members of the pedigree. The skin of the dorsal surface of the hands is not thickened although it peels off at the border of normal and keratotic skin as the photograph in Figure 3*E* shows.

Finger and toe nails are not excessively thickened in any affected members of the pedigree. No abnormalities of the joints occur, according to an X-ray examination made by R. Kegel, M.D. The fourth and fifth fingers of the propositus, IV-12, are partially flexed. Perspiration on palms and soles occurs normally in all affected individuals.

The anomaly was first observed at birth in IV-12, the propositus, as a bluish-red line encircling the areas now affected, with small, scattered white papules inside. In a sister, IV-16, the abnormality was likewise first noticed at birth, while it became apparent in a brother, IV-15, when he was forty days old.

The propositus says that he suffers no itching or tenderness on the hands or feet as a result of the keratosis. His affected sibs likewise report no inconvenience. His affected mother, III-9, although a laundress, with her hands constantly in water, claims no ill effects from her abnormality.



KERATOSIS IN TWO GENERATIONS

Figure 3

A—Condition of palms and soles of feet of the mother (III-9 in pedigree chart, Figure 5). The palms and soles of three of her children are shown in the other photographs. Details of the hands and feet of her son, IV-15, are shown at *B*, and of her daughter, IV-16, at *C*. Three detailed views of the hands and feet of her other affected son (VI-12) are shown at the right (*D-F*). Note the scaling of the skin from around the base of the finger nails (*E*).

STRATUM CORNEUM

Figure 4

Microphotograph of section of skin from instep of IV-12. The excessively thickened stratum corneum is seen above the dark Malpighian layer shown near the bottom of the photograph.

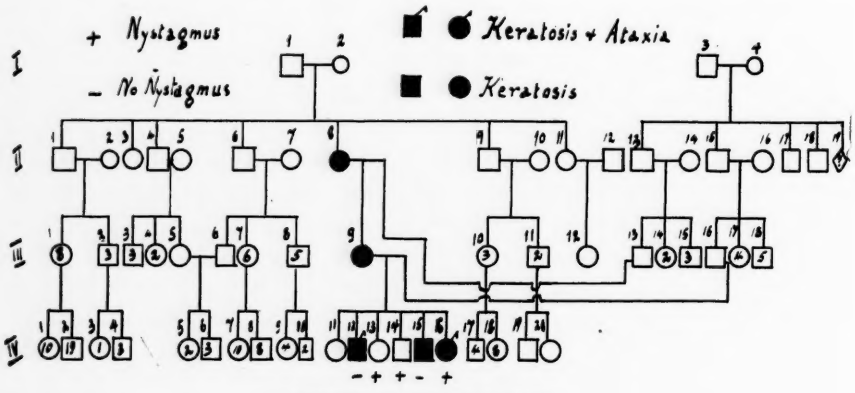
In Figure 4 appears a microphotograph of skin taken from the instep of the foot of IV-12. A study of the pathological histology of this skin has been made by Edmond Shwayri, M.D., to whom the authors owe the following description: "The epidermis is greatly thickened. The stratum corneum is responsible for the greater part of this thickening. It is about six times the thickness of all other layers combined. Small areas of parakeratosis are present. The junction of the stratum corneum with the stratum granulosum is irregular, the latter sending spurs of different lengths into the former. The stratum granulosum and stratum Malpighii are increased in thickness. Keratinized cells are seen in the latter. The sweat glands and blood vessels appear normal. There is no inflammatory infiltration in the dermis or epidermis." This description agrees with that given by Dupre and Mosny quoted by Cockayne.[3]

The sensations of touch, pain, heat, and cold were studied in the propositus, IV-12. The patient was blindfolded. The thenar eminence, the hypothenar eminence, the finger pulps, and the region at the metacarpal heads, as well as the center of the palm and the dorsum of the hand, were tested as follows: (1) A light and superficial pin prick in cornified regions produced a touch sensation but no pain. When the pin was thrust through the cornified layer for a distance of 0.5 to 1.5 millimeters, the patient showed a definite pain reaction. On the dorsum of the hand and in the region of the palmar creases, where the cornified layer is almost absent, and where the skin is red, the patient feels a sharp sense of pain from a superficial pin prick.

(2) With hot and cold water put in separate test tubes the patient was always able to distinguish hot from cold, but only after a lag period. When hot and cold stimuli were applied to the dorsum of the hand, the responses of the patient were rapidly given.

The onset of symptoms of Friedreich's ataxia were first noticed in the propositus, IV-12, at the age of ten years. Now eighteen years old, this young man has a marked ataxic gait, marked ataxic speech, marked Rombergism, absence of vibratory and deep sensation, adiadochokinesia, and general areflexia. His younger sister, IV-16, now eight years old has a mild ataxic gait, mild Rombergism, and general areflexia. In addition she has a lateral nystagmus (not spontaneous), which is not found in the propositus. On the other hand, an induced lateral nystagmus is found in an otherwise normal brother, IV-4, and normal sister, IV-13. In both sibs affected with ataxia; i.e., IV-12 and IV-16, the head is held slightly to the left side.

The pedigree of the unfortunate family is presented in Figure 5. Black circles and black squares represent women and men, respectively, affected with keratosis. Persons suffering with Friedreich's ataxia are shown with a hook at-



COMBINED MISFORTUNE

Figure 5

That two individuals in this pedigree have both keratosis and Friedreich's ataxia is probably to be attributed to chance rather than to linkage. The eye-weaving (nystagmus) appears to be unrelated to the other defects. The fact that the spouses of the two affected women were cousins would increase the likelihood that a recessive gene might appear in generation IV.

tached to the upper right corner of a circle or square. If nystagmus can be induced, a plus sign appears under the circle or square. The authors have seen III-9 and all of her children except IV-11, who died at the age of four months.

Keratosis is apparently dependent upon an autosomal dominant gene in this pedigree for reasons which will presently appear. The anomaly must have occurred as a mutation in one of the parents of II-8 since she was the only affected person in her sibship of seven persons. The abnormality appears in three successive generations of the maternal line only. The spouse of II-8; i.e., III-13, was not related to her, whereas the spouse of II-9 was the first cousin of her father. From marriages of keratotic individuals with normal individuals there were in all two affected females, two affected males, and two females and one male without the skin abnormality. Although these numbers are few, they are suggestive of the one to one ratio of affected males and females to normal individuals of both sexes expected if a dominant autosomal gene is responsible for the abnormality. According to Cockayne,[3] most cases of keratosis palmarum et plantarum show dominant inheritance.

If a recessive autosomal gene were the cause of the keratosis, III-13 would have to be presumed to be heterozygous for this rare abnormality although he was unrelated to his affected spouse, II-8. Also III-16 would have to be presumed heterozygous for keratosis although he was related to his keratotic spouse III-9 only through common ancestors I-3 and I-4 of her normal appearing father, III-13. Thus the possibility of recessive inheritance of keratosis in this pedigree, although not at all probable, cannot be definitely ruled out.

Friedreich's ataxia appears only in two offspring of III-9 and III-16. This marriage represents a slight degree of inbreeding, since III-16 is a first cousin of his wife's father. In this pedigree Friedreich's ataxia appears to be dependent upon an autosomal recessive gene since it occurs in two sibs of opposite sex out of a sibship of six from parents unaffected with the ataxia. Moreover the age of onset in the ataxic individuals was ten and eight years respectively. Bell and Carmichael[1] have shown that the mean age of onset of the recessive

form of Friedreich's ataxia is 11.7 years; of the dominant form of Friedreich's ataxia, 20.4 years. Also, the symptoms of Friedreich's ataxia are severe in the propositus, IV-12, now eighteen years of age, although they are at present mild in his youngest sister, IV-16, in whom the ataxic symptoms are just now beginning to be apparent. Bell and Carmichael[1] have shown that the recessive form of Friedreich's ataxia follows a much more severe course than the dominant form. These considerations in addition to the kinship, though remote, of the parents III-9 and III-16, point to the recessive inheritance of the ataxia in the present pedigree.

There seems to be no connection between keratosis and Friedreich's ataxia in the present pedigree. All progeny of III-9 and III-16 have now reached the age of onset of Friedreich's ataxia except IV-11 who died in infancy. Two of the keratotic individuals of this sibship were also ataxic; one keratotic individual was not ataxic. The two normal sibs, IV-13 and IV-14, showed a non-spontaneous lateral nystagmus which possibly indicates heterozygosity for the recessive gene causing ataxia. In no previously described case has there been an association of keratosis and Friedreich's ataxia.

The authors are indebted to Dr. Alan C. Pipkin for assistance in securing the photographs and Dr. Curtis Baylor, M.D,. for neurological examination of the ataxic individuals.

Summary

A pedigree is described in which both Keratosis palmarum et plantarum and Friedreich's ataxia occur in certain members of a sibship. This keratosis is apparently dependent upon an autosomal dominant gene; Friedreich's ataxia, on a recessive gene. There is probably no connection between the two abnormalities in this pedigree.

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FEULGEN FOR FULBRIGHT (AND SEE CHINA TOO!)

There is an opening for a biologist of instructor or assistant professor level to teach cytology and genetics at National Szechuan University, Chengtu, China for this coming academic year. The opening is in connection with the United States Government educational exchange program (Public Law 584, the Fulbright Act). Inquiries should be addressed to the Executive Secretary, Conference Board of Associated Research Councils, Committee on International Exchange of Persons, 2101 Constitution Avenue, Washington 25, D. C.

REPORT OF OCTUPLETS AND SEXTUPLETS IN THE UNITED STATES

WHEN in 1937 I was gathering material for my book, *Multiple Human Births,* I read all available records about the frequency of occurrence of twins, triplets, quadruplets, and larger sets. At that time it seemed safe to say that "Multiple births consisting of as high as six babies at a time actually have occurred, but it is highly improbable that higher numbers at a birth have ever occurred. . . . Only six cases of sextuplets have been reported in the medical literature, and of these only four are regarded as authentic." I have also repeatedly stated in lectures that there was no reliable record of any sextuplet or higher-numbered set born in the United States, and that there were only four sets of quintuplets.

During the past few weeks, however, I have had correspondence with Mrs. Bert Tanner of Lodi, Wisconsin, who has been interested for some time in multiple human births and has collected some data which may cause me to retract my former statements.

A Case of Octuplets in Ohio

Mrs. Bert Tanner has in her possession a clipping from *The Columbus Democrat,* published at Columbus, Wisconsin, on September, 1872, copied from the *Cincinnati Lancet* for August, 1872, that reads as follows: "On the 21st of August, Mrs. Timothy Bradlee of Trumbull County, Ohio, gave birth to eight children, three boys and five girls. They are all living and healthy, but quite small. Mr. Bradlee was married six years ago to Eunice Mowery, who weighed two hundred and seventy-three pounds on the day of her marriage. She has given birth to two pairs of twins, and now eight more, making twelve children in six years. Mrs. Bradlee was a triplet, her mother and father both being twins, and her grandmother the mother of five pairs of twins."

If this is an authentic account, it means that eight babies were born at one delivery and that all were living one month later. This would put the case of the Dionne quintuplets very much in the shade. I have no way of checking this case, but perhaps its publication in this JOURNAL will throw some light on it. There may be some persons who know what sort of journal the *Cincinnati Lancet* was, or there may be some one who knows of the Bradlee family and may be able to tell us about the case.

Until the case can be authenticated, if it ever can be, it would be futile to speculate about its implications. Yet one must admit that the account is circumstantial enough to bear the stamp of authenticity. It would seem to be a case of a sort of inbred twinning family, possibly homozygous for the genetic factors favoring multiple births. More than that I, as a twinning specialist, would be unwilling to say.

The Chicago Sextuplets

Mrs. Tanner also sent me a page from *Look* magazine of October 25, 1938, giving an account of the birth of sextuplets, together with photographs of three of the members of the set with their names and addresses. There is also a photostatic copy of the birth certificate written by the attending physician and signed by him and the midwife and nurse. The certificate reads as follows:

"Chicago, Sept. 15, 1866
This is to certify that Mrs. Winnie A. Bushnell wife of J. L. Bushnell in the City of Chicago on the eighth day of September 1866 gave birth to six living children three boys and three girls.

James Edwards, M.D., attending physician, Priscilla Bancroft, midwife and nurse.

Look magazine has the following comments about the case. "A Doctor's written statement is the only testimony to the birth of the Bushnell sextuplets. Chicago authorities state that birth rec-

ords prior to 1871 were burned in the great fire. Although the phenomenal Bushnell births are impossible to verify through official sources, persons who knew the family at the time of the event confirm the story of sextuplets."

Beneath the photographs of two of the survivors of the set is the following legend: *"More Unusual Than the Dionnes,* this elderly pair are two of the sextuplets whose birth in Chicago was kept a secret for 72 years. They are Alberto Bushnell of Albion, N. Y., and Mrs. Alinca Parker of Silver Lake, N.Y. A sister, Mrs. Alice Hughes lives in Arizona and a brother, Norberto, died at 68. Another brother, Loberto and sister Amelia died at 8 months. The marvelous birth was kept secret because 'Mother didn't think sextuplets were anything to be proud of and father seemed to agree,' Mrs. Parker explains."

Beneath a full-length photo-engraving of Mrs. Hughes is the following legend: *"The Third Surviving Sextuplet* is Mrs. Alice Hughes. This picture was taken in 1923 at her Flagstaff, Arizona, home, where she resides with her husband. She and her brother and sister were discovered by Robert Ripley, 'Believe It or Not' creator. The latter two were recently guests of Ripley's radio broadcast."

It seems to me that this case of sextuplets is about as nearly authentic as any case as old as this is likely to be. For myself, I am willing to withdraw my claim that there has never been a true case of sextuplets in the United States.

The alleged case of octuplets all born alive and all living for at least a month is much more difficult to accept. Yet it cannot be ruled out arbitrarily because it is unique and very improbable. It ought to be possible to ferret out some further evidence either in support of the claims made in the *Cincinnati Lancet* or to establish that the story is a fake. It is with the hope of learning something further about "the Bradlee octuplets" that this paper has been written.

If anyone has any information concerning any aspect of these cases, or of other similar cases, please write to me, care of the editor of the JOURNAL OF HEREDITY.

H. H. NEWMAN

Clearwater, Florida

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First International Congress of Animal Reproduction

On Wednesday, June the 30th, took place the closure meeting of the 1st International Congress of Physiology and Pathology of Animal Reproduction and Artificial Insemination, which was held in Milan under the auspices of the University and of the Institute "Spallanzani" and which was opened on the 23rd of June with participation of the representants and delegations of 35 countries from all continents. About 600 workers took part in the Congress of which 300 came from abroad. Over 250 papers were presented which were divided into 5 sections. During the Congress also various other scientifical meetings were held. Besides, wares shows and livestock exhibitions took place. In the latter, animals of the leading Italian breeds as well as livestock from Canada, France, England, Holland, Spain, Switzerland, and from Voralrlberg were exhibited. The Congressists also made excursions to various interesting places.

The Congress has accepted the the following final resolutions: it has recognized the importance of close collaboration among the workers of all sciences and has recommended the elaboration of an organic mutual program to be extended nationally and internationally as well; it has recognized the importance of scientific meetings and international congresses to be held rather frequently; and it has decided to name a Permanent Committee for Physiology and Pathology of Animal Reproduction (including artificial insemination) who have also the task of organizing within three years from now, the 2nd International Congress of Physiology and Pathology of Animal Reproduction and Artificial Insemination; finally it has recommended the adoption of the abbreviation A.I. to express artificial insemination.

The above permanent committee is composed of the following 9 members: Prof. J. Anderson (Kenya), Prof. T. Bonadonna, Secretary-General (Italy); Prof. E. Garcia Mata (Argentina); Prof. R. M. C. Gunn (Australia); Prof. J. Hammond (England); Prof. N. Lagerlof, President (Sweden); Prof. G. Lesbouyries (France); Prof. B. B. Morgan (U.S.A.); Prof. E. Sorensen (Denmark).

INHERITANCE OF FRONTAL HAIR DIRECTIONS IN MAN

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THE hair of mammals as a rule forms an oblique angle with the skin surface. In this way the sloping hairs make up hair streams which follow more or less definite directions. Descriptions of the different hair stream patterns of the human body have been published by a number of workers.†

Various theories have been advanced to account for the existence of hair direction patterns. Landauer[11] suggested that the sloping direction of mammalian hairs is determined by forces of mechanical stress during development and that the degree of slope is influenced by such physical factors as the thickness of the skin and of the hair. This view has been the subject of studies by Trotter and Dawson[24] on rats, by David[5] on mice and rats and by Upham and Landauer[25] on human scalps. Niedoba[14,16,17] contributed to the analysis of these problems by describing abnormalities of hair direction in animal monsters. Gastberger[8] found a structural connection between the direction of hair and the course of the septum system of the subcutis in human fetuses and infants. Blechschmidt[2] observed a slanting latero-caudal directed growth movement in the outer stratum of the guinea pig embryo and supposed that this was part of a uniform growth movement in the embryo, originated excentrically somewhere in the head area. Schönherr[22] examined the histology of human scalp whorls and found that the direction of hairs was due to an outward directed growth movement of the skin.

Very little is known about hereditary factors influencing or determining the origin of these hair streams in man. The only investigations concerning the heredity of human hair direction which I have been able to find in the literature, refer to the whorls at the top of the head. Brewster[3] published a pedigree showing the occurrence of double crowns in three successive generations. Bernstein[1] found that the clockwise direction of the crown whorl was a monohybrid dominant hereditary character. Snyder[23] also arrived at the conclusion that the different directions of the occipital hair-whorls are determined by a pair of unit factors. He found that these factors were linked neither to the blood-group factors nor to eye color. Examinations of the occipital whorls were also made by Lauterbach and Knight[12] on schoolchildren and on feebleminded individuals. Verschuer,[26] adding his own observations to those of Newman, records 31.3 per cent of discordance in whorl direction among 163 monozygotic twin pairs.

In regard to the genetics of hair whorls in animals, the well known work of Castle[4] and Wright[29,30] on guinea pigs should be mentioned. Wright found that the rough fur type differed from the smooth wild type by two major factor pairs. The one is Castle's *R* which is necessary for any development of this type of roughness. The other, *m*, is an incomplete recessive necessary for full development of the character. Pictet and Ferrero[20,21] postulated three different dominant genes producing hair whorls

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†Osiander[19], Eschricht[6], Voigt[28], Kidd[9,10], and Ludwig[13]. After this paper had been sent for printing, I have had the pleasure of reading L. Bolk's important description: On the Hair Slope in the Frontal Region of Man (*Journ. Anat.* V. 58:206-221, 1924). The near congruity between Bolk's observations on human fetuses and this author's on children and adults is striking.

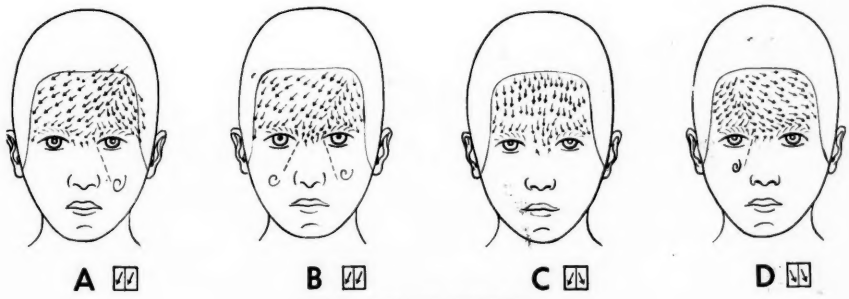


Figure 6

In Type I the direction of the hair streams on the forehead is downward, with a frontal convergence line located at the eyebrows. See text for details.

in the skin of guinea pigs: a dominant gene *R*, acting on the head and trunk; another gene, *D*, acting only on the head hair and producing fully developed rosettes, while the recessive allele, *d*, is said to be responsible for rudimentary rosettes. Pictet and Ferrero also postulated a third dominant gene, *G*, necessary for the formation of hair rosettes on the trunk.

Niedoba[14] found that double frontal whorls in horses showed no dominance and that double whorls in both parents resulted in double whorls in the offspring. In swine Nordby[18] reported inherited whorls in the hair, mostly in the region of the neck, withers and rump. He stated that the inheritance was not simple.

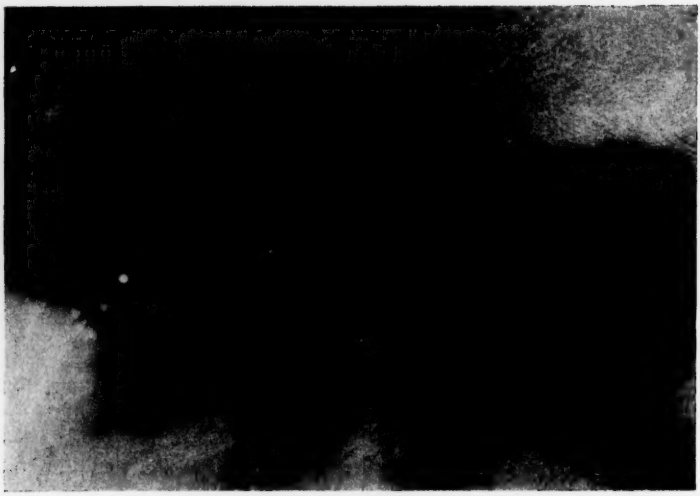
Material and Methods

In order to gain some knowledge about the inheritance of the different directions of the hair streams in the frontal region of man, the author has collected material on Norwegian children, twins and families.

In man the original fetal hairs (the lanugo) are replaced by the secondary hairs. On the head, the secondary hairs are of two kinds: thick, coarse and long hairs (capillus) on the scalp, beard and eyebrows, sometimes called terminal hair; and short, delicate down hairs (vellus) on the forehead and other parts of the face, but also found in large numbers on the scalp.

The direction of the capillus is very easily observed, usually throughout life. For observation of the direction of the down hair it is most convenient to use the light from an electric flashlight. When the light is projected in the hair direction, the down hairs can be observed with facility. If the light is projected in the opposite direction (from the tip towards the root), the down hairs are difficultly visible. For minute observations a magnifying glass is used.

The hair streams of the vellus are more distinct in children than in adults and more distinct in adult females than in adult males. With increasing age there are some alterations in hair direction, especially that of the vellus, resulting in reduced slope of hairs. This has probably something to do with the thickness of the skin and accords with the results obtained by Frédéric[7] and by Upham and Landauer.[25] By studying the human scalp they found significant positive correlations between thickness of cutis and angle of hairs with the cutis as well as between combined cutis and subcutis thickness and angle of the hairs with the cutis. In very old persons there often seems to be some atrophy of the follicles of the vellus; in such cases it is very difficult to determine the direction of the vellus.



TYPE I HAIR CROSSES

Figure 7

The eyebrow region of a young woman showing two frontal crosses in the eyebrows, a common situation caused by "colliding" hair streams.

The Different Types of Frontal Hair Direction

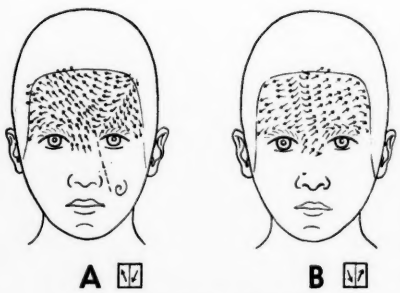
In the frontal region of man two hair streams meet: the one, here named the crown stream, originating at the crown, and the other, the glabellar stream, starting from the root of the nose. The meeting line of these two streams is here called the frontal convergence line. The crown stream often shows a more or less distinct frontal divergence line, usually lying on the left side. If the two streams meet at an angle of about 180°, they will form so-called crosses (shown in Figures 6*A*, 6*B*, 6*D*, 8*A*, 9*B*, 9*D*).

The frontal convergence line between these two streams has a very variable position. In spite of this practically all of the individuals investigated could be placed definitely in one of the following three groups:

Type I (Figures 6*A-D*) shows downward hair direction on both sides of the forehead and the frontal convergence line located at the eyebrows. Type I characteristically forms hair crosses near the root of the nose, and in one (usually the left) or both eyebrows (Figure 7).* In most persons the divergence line of the crown stream lies on the left side, and these individuals usually arrange their hair with the parting on the left (Figures 6*A, B*). In some cases the divergence line is not so pronounced, and the hairs are growing in more nearly parallel directions on the forehead (Figure 6*C*). Sometimes the divergence line is found on the right side (Figure 6*D*).

Type II: (Figure 8*A, B*) the hair direction is downward on one, usually the left, side of the forehead and upward on the other, that is, the glabellar stream runs from the root of the nose upward towards the right, pressing the crown stream more or less backward on this side of the forehead. The frontal con-

*Virchow[27] reported the frequent occurrence of a "whorl" in the eyebrow in a particular family; the illustrating photograph seems to show a case of the common type of frontal crosses in the eyebrow.



TYPE II HAIR STREAMS

Figure 8

In Type II we have a combination of downward and upward hair streams variously arranged. The downward stream is usually on the left, but not always.

vergence line is in this way situated above the eyebrow on the right, while on the left side the crown stream reaches the eyebrow (Figure 8*A*). The inverted type (Figure 8*B*) is very infrequent.

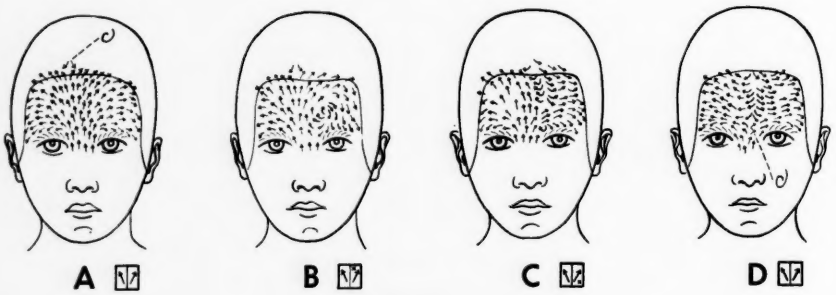
In *type III* (Figures 9*A-D*) the glabellar stream runs more or less symmetrically upward, spreading fanwise on both sides of the forehead. The frontal convergence line is situated above the eyebrows on both sides. As a rule the glabellar stream reaches or crosses the border of the scalp (trichion) (Figures 9*A, 9B*); in some cases the border is reached only on the right side (Figure 9*C*), in others the frontal convergence line is found between the border and the eyebrow on both sides of the forehead (Figure 9*D*). Frontal hair whorls are found relatively frequently in connection with this type (Figure 9*B*) but will now and then be found also with the other types. In practically all individuals of type III the glabellar stream is directed a little more to the right, causing the frontal convergence line to lie somewhat higher on that side.

Frequency of the Different Types In A Norwegian Population

Table I shows the distribution of the different types of hair direction on the forehead in 200 schoolboys 6 to 8 years old in Oslo, Norway. Eighty-five of these, or 42.5 per cent, showed type I, 70 of them having the divergence line on the left side. Type II and type III had the same frequency in this material, each of them having been found in 56 individuals or 28 per cent. Of type II, 49 boys had the divergence line on the left. In 45 of the boys belonging to type III, the convergence line reached or crossed the border of the scalp on both sides. Only three of the 200 individuals could not with certainty be placed in any of the three groups, owing to the formation of strong abberant whorls on the forehead of two of them, the third boy showing large scars in the scalp caused by obstetrical forceps at birth. The hair pattern of this last mentioned was very irregular. An investigation of 85 other young Norwegians gave a similar result (Table II). 44.7 per cent belonged to type I, 27.1 per cent to type II and 28.2 per cent to type III. Of this group 37 were girls, 48 boys. The material was collected in secondary schools, hospitals and an infant

TABLE I. Frontal hair directions in 200 school boys of Oslo, 6-8 years old.

	N	%
Type I: Downward hair direction on both sides. Frontal convergence line in the eyebrows.		
Divergence line:		
on the left, no frontal whorl	66	
on the left, with frontal whorl	4	
on the right, no frontal whorl	6	
on the right, with frontal whorl	1	
at the median	6	
not distinct	2	
Total	85	42.5
Type II: Upward hair direction on the one side, downward direction on the other. Frontal convergence line coincides with the eyebrow only on the one side, and is situated above the eyebrow on the other.		
Upward direction towards the right:		
Divergence line:		
on the left, no frontal whorl	35	
on the left, with frontal whorl	14	
on the right, no frontal whorl	3	
on the right, with frontal whorl	1	
Upward direction towards the left:		
Divergence line at the median	3	
Total	56	28
Type III: Upward direction on both sides. Frontal convergence line above the eyebrows on both sides.		
Convergence line reaching the border of the scalp:		
on both sides, no frontal whorl	35	
on both sides, with frontal whorl	10	
on the right, no frontal whorl	4	
on the right, with frontal whorl	3	
Convergence line below the border of the scalp on both sides:		
no frontal whorl	4	
Total	56	28
Undetermined, aberrant	3	1.5
Total	200	100



TYPE III HAIR STREAMS

Figure 9

In type III, the hair streams are directed upward from the region of the eyebrows toward the forehead. Important subgroupings of this type are shown here.

home. There was a specially large number of girls of type III, but this seems to be of no significance, the material being too limited for such observations.

One-Egg and Two-Egg Twins

The comparative investigation of one-egg (monozygotic) and two-egg (dizygotic) twins is the best way of determining if a charater is due to hereditary factors and to what degree modifications are induced by the environment.

The material here published consisted of 30 pairs of one-egg and 28 pairs of two-egg twins. The one-egg twins (Figure 10, left side) showed complete concordance in relation to the three types of frontal hair direction. In the two-egg twins (Figure 10, right side) only 43 per cent showed concordance and 57 per cent were discordant in relation to the type of hair direction. But even among the 43 per cent of concordant twins there was a higher degree of difference in the hair arrangement between the two partners than between the partners of one-egg twins. Slight differences were found among the one-egg twins, especially with respect to the height of the frontal convergence line in type III. The most striking case is presented in Figure 11 which shows a pair of one-egg twin boys both of type III. Both of them had a whorl at the frontal convergence line on the left side, but in one of them the whorl was situated just at the border of the hair line, while the other one had the whorl a little (c. 1 cm.) below the border. — In the one-egg twins there also occurred mirror-imaging with respect to the type of frontal hair direction.

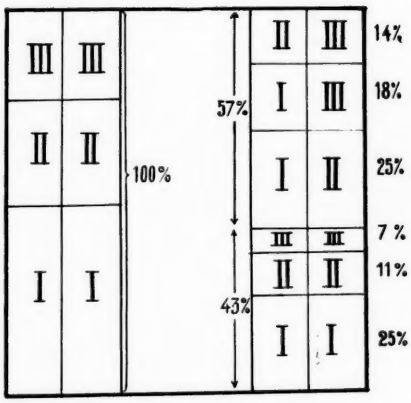
TABLE II. Frequency of the different types of hair direction in 85 young Norwegians.

Type	I		II		III		Total	
Sex.	♀	♂	♀	♂	♀	♂	♀	♂
n	12	26	8	15	17	7	37	48

The Family Material

Family material is best suited for the analysis of such hereditary factors. For this reason I have examined a considerable number of families of which only the most illustrative pedigrees are commented on here.

Figure 12*A* represents a family where the mother is of type III, the father of type II. Two of the six children are of type III like their mother, three of type II like their father, but one of the children shows type I, not present in either of the parents. In Figure 12*B* the mother is type II, the father type III, with a frontal whorl on the left side (whorls in the vellus are difficult to observe in adults). Of the six children examined, two show their father's type, both with frontal whorls, two others have the mother's type and the remaining two are



HAIR STREAMS IN TWINS

Figure 10

On the left are shown frequencies of concordances in identical twins, which in this group are 100 per cent. On the right are shown the concordance frequencies of 28 pairs of two-egg twins. Of these 57 per cent were discordant. The majority of the concordant two-egg twins had Type I hair patterns.

type I, both of the ordinary type, with the divergence line on the left side. The same sort of pedigree has been found in other families, indicating that type I probably is recessive to both type II and type III. This is also confirmed by pedigrees like that of the family shown in Figure 12*C*, where both parents are type III; among their seven children, two show type I, the other five type III, indicating that their parents, like those mentioned before, probably are both heterozygous for type I.

In the next family, Figure 12*D*, where also both parents belong to type III, four out of ten children are type II. The probable explanation is that neither of the parents is homozygous for type III, but that one or perhaps both carry the factor for type II and that this type is hypostatic to type III. A similar case is found in Figure 12*E*, in a family with one-egg triplets. Of the seven children all the triplets and two others are of type III like both their parents, while two of the children are type II.

In the family of Figure 12*F* the mother is type I, the father type III. Of 11 children only 9 were examined. Six had the type of their mother, three the type of their father. Photographs of the eldest daughter showed that she probably was type III (double contour in the diagram). In this case the father presumably is heterozygous for type I. This family had two pairs of two-egg twins, both showing discordance with respect to the type of frontal hair direction.

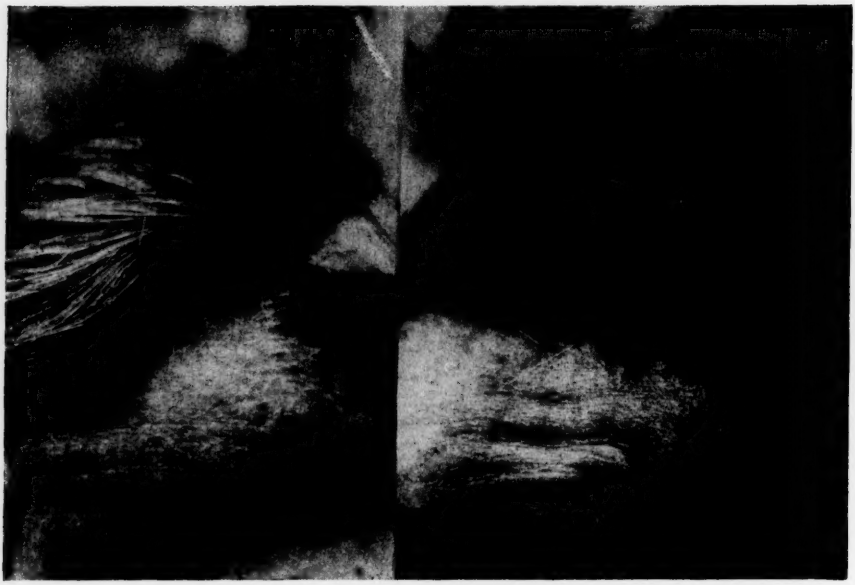
The family represented in Figure 12*G* shows the mother to be of type II, the father of type I with the divergence line located nearer the median line. Here the mother probably is heterozygous for type I: six children out of nine are of type I (three have the same type of divergence line as their father (three have the ordinary type); the other three have the dominant type of their mother.

Figure 12*H* shows a pedigree with the mother of type I and the father of type III. Of the six children, two had type III and four type II; since none had type I like their mother, the father probably had no factors for this type.

In the pedigree presented in Figure 12*I* at least one of the parents of the last generation probably is homozygous for the factor producing type III, since all the eight children have this type. (Double contour in the diagram means that the type is determined from photographs. Unfortunately it is mostly type III, which only in lucky cases may be determined in that way.)

In the family of Figure 12*J* the mother is type II, the father type I, but, in contrast to the family in Figure 12*G* all their eight children are type II, indicating that the mother probably is homozygous for this character. Four of the children, however, differed from the other four in having the convergence line located nearer the right eyebrow; this may suggest the different effect of the two gene partners in the mother.

Of cases where both parents are of type I, there are unfortunately in this material only small families of children, the largest containing five examined children, all of type I. In Figure 12*K* is



HAIR WHORLS IN TWINS
Figure 11

One-egg twins of Type III are concordant as to general pattern but discordant as to details. The twin on the left has a whorl just above the trichion (hair line on forehead). His co-twin has a whorl just below the trichion. The dots show the location of the whorls.

shown another family of the same type in three generations. In these cases both the parents are supposed to be homozygous for type I and without the factors producing any of the other types. All children of such marriages are supposed to be type I. In the twin material, 11 families in all were examined (5 with one-egg twins and 6 with two-egg twins) with both the parents of type I. All the 29 children (including the twins) showed type I.

In Figure 12*L* the parents of the first generation (left side) both are type I and so are both their two examined children. Both children married persons of type III, the woman involved (right side) was probably homozygous for this type. Both children of this marriage have type III and so has the child of the other marriage.

Some additional pedigrees, not discussed in detail, and some additional data regarding twin sibships and small

TABLE III. Small families with twin pairs. *MT* = monozygotic twins, *DT* = dizygotic twins. I, II and III are the different major types of frontal hair directions.

No.	Father	Mother	Children					
			I		II		III	
			♂	♀	♂	♀	♂	♀
1	I	I	2 MT 1					
2	I	I		2 MT				
3	I	I	2 MT 1					
4	I	I	2 MT					
5	I	I	2 DT					
6	I	I	2 DT					
7	I	I	2 DT					
8	I	I		2 DT				
9	I	I	1 DT	1 DT 1				
10	II	II		1 DT		1 DT 1		
11	III	III					2 MT	1
12	II	I			2 MT	1		
13	III	I	1 DT					1 DT
14	I	III		2 MT				
15	I	III	1				2 MT 1	
16	I	III		2 MT				
17	I	III	1				2 MT	
18	I	III					2 DT	
19	I	III	2 DT					
20	III	I				1 DT		1 DT 1
21	II	III					2 MT	
22	II	III			2 DT 1			
23	III	II				1 DT		1 DT
24	II	III				2 MT		

families are presented in Figure 13 and Table III.

Discussion

A summary of the pedigree data indicates the following:

In all cases where an individual showing type III was found, either one or both of the examined parents were of type III, indicating the dominance of this type.

In all cases of an individual showing type II, both examined parents were never of type I; in these cases the parents were either II × I, II × II, II × III, I × III or III × III, these last two combinations indicating that type II is recessive to type III.

In all cases where the parents both were type I, all their children were of the same type.

If one of the parents showed type I and the other type III, the material had no case of all three types in the children.

Consequently, in no single family in the whole material, containing 19 large families (with more than five children) and a larger number of small families, is there found evidence conflicting with the assumption that these three types are produced by allelomorphic genes. Of course, there is the possibility of close linkage; this seems, however, less probable.

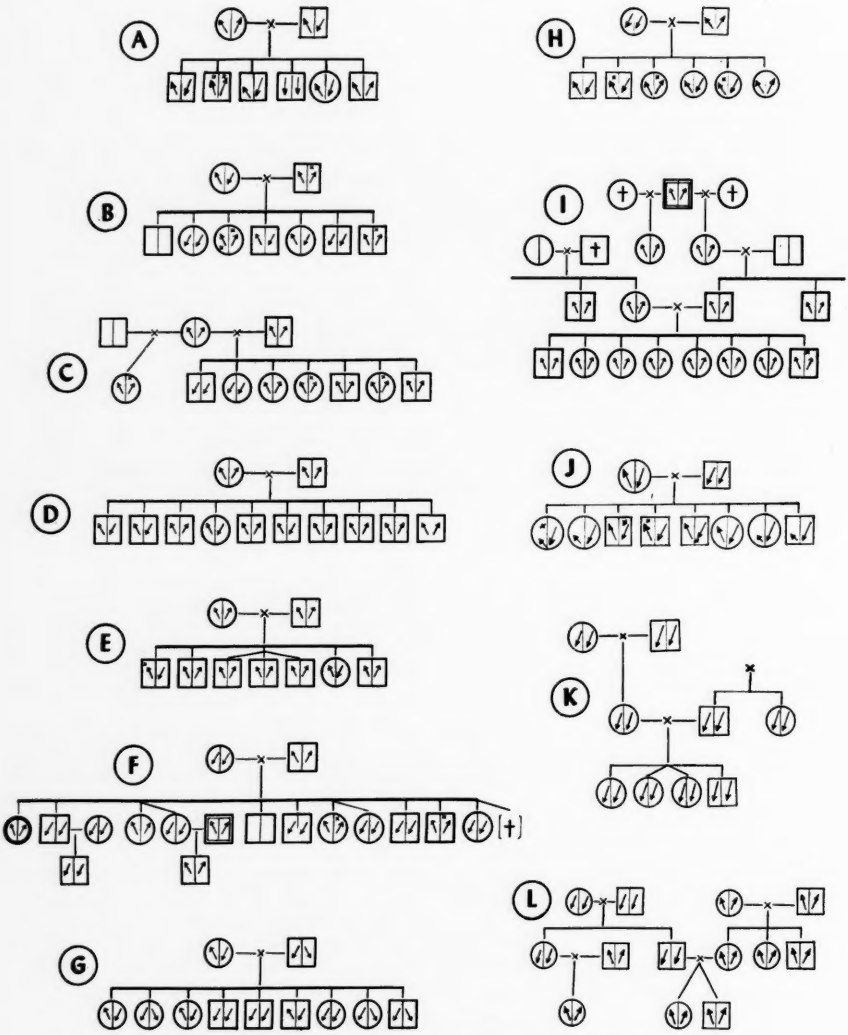
Further it will be observed from the pedigrees that the family data give no support to the supposition of sex-linkage in any of these types. In reality they are quite contrary to such a theory.

The material seems to indicate that the different types of frontal hair direction in man are brought about by autosomal multiple allels. The genes producing the three main types (type I, type II and type III) therefore may be named d^{I}, D^{II} and D^{III}, respectively. Individuals with (pheno) type I then are supposed to have the genotypic constitution $d^{I}d^{I}$, those with (pheno) type II either $d^{I}D^{II}$ or $D^{II}D^{II}$ and those with (pheno) type III either $d^{I}D^{III}$, $D^{II}D^{III}$ or $D^{III}D^{III}$.

These genetic factors presumably are of growth-regulating nature, probably with an effect located in the region of the root of the nose and active only in a particular stage of embryonic development, most likely coincidental with the first formation of the lanugo hairs in the frontal region. It seems probable that local differential growth tensions in the different layers of the skin may determine the slope of the hair. Thus the largest pressure of growth will produce the largest extension of the glabellar stream, type III. The formation of the different types therefore probably are the result of a competition between the coincidental growth intensities of two embryonic growth localities, the one in the area of the root of the nose and the other in the area of the occipital vertex, the factor d^{I} giving a minimum of growth intensity in the glabellar area and D^{III} the maximum. This hypothesis needs to be verified by experiments.

The variations within each of the major types in this material is considerable and may have different causes. The variations may conceivably be due to two kinds of genetic variants: either in the nature of minor allelomorphic gene series within the major d^{I}, D^{II} and D^{III} (f. ex. D^{IIa}, D^{IIb}, . .) or in the nature of inherited modifiers, or in both.

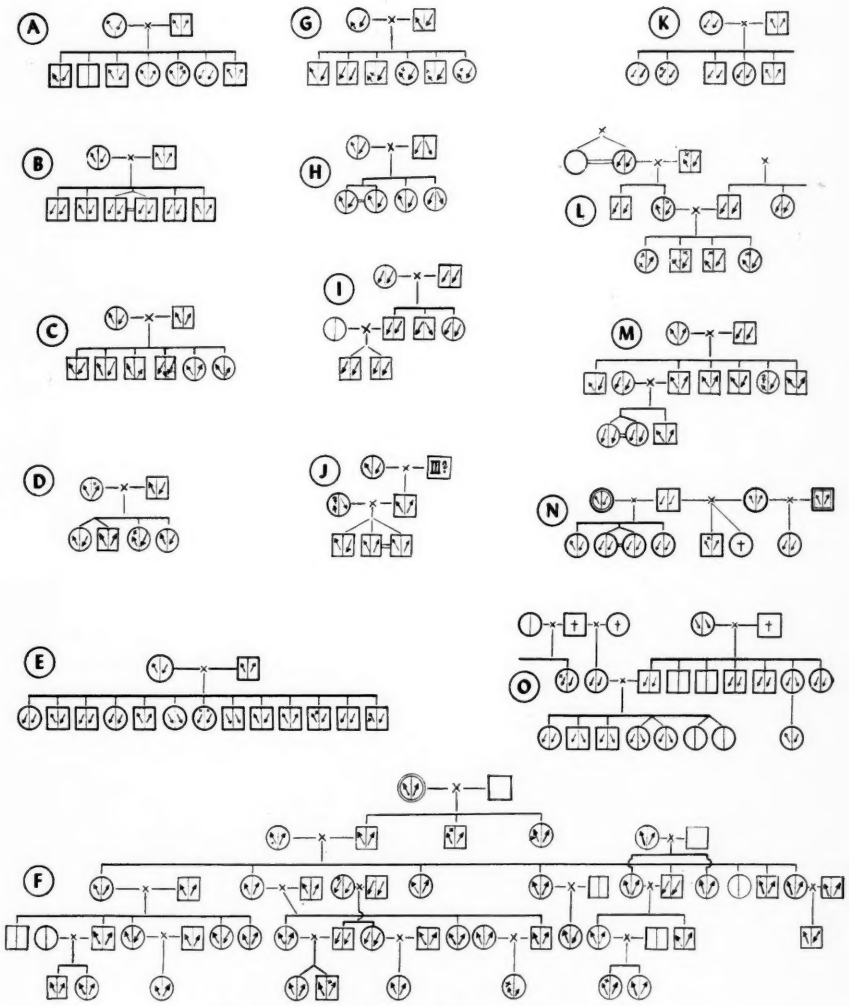
Further it is probable that the embryonic environment has some responsibility for the detailed shape of the hair streams, this being the presumable explanation for the different localizations of frontal whorl in the one-egg twins mentioned in the description of Figure 11. In this connection there could be considered such fetal influences as an eventual difference in the nutritional conditions, causing dissimilarity in the growth rhythm of the two fetuses. The different uterine position of the fetus may also have some physical influence on the growth of the fetal skin. For areas like the neck this seems to be probable and may explain the frequently found discordances there; for the frontal region, this, however, seems less probable.



See the text for description of symbols and discussion of individual pedigrees.

HAIR PATTERN PEDIGREES

Figure 12



Additional pedigrees not discussed in the text.

HAIR PATTERN PEDIGREES

Figure 13

Summary

Three different major types of frontal hair direction in a Norwegian population are described. The frequencies of these types in the material are about 44 per cent of type I, about 28 per cent of type II and about 28 per cent of type III.

The twin material shows 100 per cent concordance in one-egg twins and only 43 per cent concordance in two-egg twins.

The family material indicates that these three types probably are produced by three autosomal allelomorphic genes, d^{I}, D^{II} and D^{III} (D^{II} is recessive to D^{III}). It is suggested that these genetic factors are of growth-regulating nature, probably with an effect located in the region of the root of the nose and active in a stage of the embryonic development coincidental with the initial formation of the hair primordia.

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